radial carpal (scaphoid), is subjected to particularly severe compression forces, and in addition to arthritic changes this bone is commonly the seat of stress fractures, pathological specimens almost invariably showing severe bruising of the soft tissues within the bone (Rooney 1963). Although it is widely assumed that the site of lesions in a joint is the one exposed to the greatest stress of weight-bearing it has been suggested that injury to the cartilage by the disturbance of fluid permeation and hence nutrition is a more important factor than the mechanical stress (Harrison *et al.* 1953).

Posture: It has long been known that abnormal skeletal stresses arising from postural and structural abnormalities can induce pathological changes in young, healthy cartilage. Thoroughbreds are particularly prone to poor conformation which is often inherited. Whether the inheritable factor regarding the susceptibility of the thoroughbred to arthritis is purely one of conformation or whether there is a further genetical basis similar to the 'arthritis-prone' and 'arthritis-resistant' strains of mice reported by Sokoloff poses an interesting question (Sokoloff *et al.* 1960).

Nutrition: Unknown dietary deficiencies (Mitchell 1937, Kelser & Callender 1938), vitamin A deficiency (Howell *et al.* 1941), and trace element deficiencies have all been reported as the cause – direct or indirect – of arthritis. However, it is the role of calcium and phosphorus metabolism in joint disease that has received the most attention since it was found that either a deficiency of calcium or an imbalance in the calcium/phosphorus ratio could cause osteomalacia with accompanying joint lesions in the horse (Kitner & Holt 1932). However, the histopathology of equine osteomalacia is that of osteitis fibrosa and quite different from that of degenerative or inflammatory osteoarthritis. Furthermore, in recent work on the condition in which the bone changes were shown to be due to secondary hyperparathyroidism, no joint lesions were observed (Krook & Lowe 1964). However, many of the clinical features characteristic of osteitis fibrosa in the horse, such as stress fractures and the tearing of ligaments at their point of attachment to bone, are commonly seen accompanying traumatic arthritis in the racehorse. It is also true that young thoroughbreds often receive rations that are low in calcium and with a calcium/phosphorus imbalance. Since the last war, the feeding of higher protein diets to weanlings and yearlings together with the sometimes overzealous use of proprietary 'shot-gun' mineral-vitamin supplements has become popular and appears to have coincided with an increased incidence of enchondroses such as epiphysitis as well as arthritis.

Conclusion

Our knowledge of traumatic arthritis in the young thoroughbred is still superficial. Important though the effects of wear, tear and injury undoubtedly are, much deeper underlying factors are probably involved in the production of this disease.

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Dr W S C Copeman (*London*) said that before the last war he had been provided with facilities for studying every case of arthritis which occurred at the London Zoo. Osteoarthritis was commonly found in the heavy wild animals which were limited in their cage space, especially bears. It had been noted that the cartilage lesions were not always situated at the areas of maximum pressure, and that cartilage everywhere appeared to show a limited capacity for repair even in old animals. It had also been noted, as the result of X-ray studies, that the trabecular architecture of the weight-carrying bones was of importance in determining certain lesions. In some cases local blood-vessel infarction seemed to initiate aseptic necrosis or degeneration within the region supplied by it.

He suggested that in investigating the viscosity of joint fluid insufficient attention had been paid to hyaluronic acid metabolism, and mentioned that in the field of human osteoarthrosis the belief was growing that the basic defect might lie in a diminished capacity of the chondrocytes to produce sulphated mucopolysaccharides, thus leading to excessive loss of chondroitin sulphate.

Dr Copeman felt that it was difficult to see from the published experiments how the effect of the various procedures on animal arthrosis could constitute other than relatively nonspecific influences upon growth and metabolism. This would tie up with the failure to demonstrate in human cases any clear relationship between endocrine or chemical abnormalities and the progress of the disease.

Speaking of rheumatoid arthritis he stressed the need for a comparable experimental form in animals if further progress in knowledge was to be achieved. He pointed out that at present arthritis produced in animals was not self-perpetuating.